is constant.[3] Recent data indicate a considerable increase in the incidence of neonatal rubella in Britain in 1982,[4] which has already been reflected in the number of children with congenital rubella reported to the southern registry of the National Congenital Rubella Surveillance Programme (W C Marshall, personal communication).

All this suggests that our immunisation policy needs review, and figures for immunisation in the last five years in the Brent health district may have some messages.[5] While an average of 85% of infants completed primary courses of immunisation against diphtheria, tetanus, and polio, only 45% were immunised against petussis and only 39% of children completed a primary course of immunisation against measles. Since 1970 the Department of Health and Social Security has followed a policy of immunising girls between the ages of 11 and 13 against rubella, and uptake was expected to be about 70%. This degree of compliance has been achieved in Brent—but that necessarily means that one third of women and two thirds of the total population remain as a reservoir of infection. Clearly we are reaping the consequences of our own inaction, so what can and should be done?

Firstly, with health visitors closely in touch with young families it should not be difficult to increase immunisation rates against diphtheria, tetanus, and polio. Health workers should be clear that the risk of sustaining brain damage from natural pertussis is several times greater than the risk of sustaining brain damage from the immunisation. This message should be widely propagated among clinic doctors and nurses, and every effort should be made to increase the uptake of what is demonstrably an effective prophylactic treatment. Official advice on the postponement of immunisation if the patient is suffering from any acute illness should be reconsidered.[6] It is my experience that very minor snuffles may indefinitely postpone the immunisation of just those children who are most in need of it. Defensive medicine may be taken too far, and a competent doctor ought to be able to spot whether or not a child is clinically ill, which is the only real contraindication to proceeding.

If the clinic shows an active interest in immunisation procedures, especially towards the first child, the chances must be greatly increased of that child receiving measles vaccination at the end of the first year and of subsequent children also being immunised. Whether we should move towards compulsory immunisation as a precondition to school entry (as currently practised in the United States) may be debated, but child health registers and the system of primary care in Britain ought to be equal to the task of matching the United States Public Health Service, which has set as its goal the early eradication of measles.[7]

No scientific defence is possible of the current British approach to rubella vaccination.[8] It has failed to protect women of childbearing age, with immense costs in human terms let alone in the provision of services for handicapped children. This year should see a redirection of our policy, designed firstly to protect women of childbearing age and then to interrupt the transmission of rubella and eradicate the disease. Implementation of the first priority entails vaccinating all women and girls of reproductive age and the second requires that all children currently aged 1-14 should be vaccinated, thereafter vaccinating all children at 1 year. There is no alternative to this policy in a caring society, and doctors have a duty plainly to tell these facts to the nation.

Finally, several newer vaccines are available but have been ignored. There has been little discussion of mumps immunisation in Britain, and, though the benefit of such immunisation might seem to be marginal, it is not for the patient who develops mumps orchitis, pancreatitis, or diabetes. Pneumococcal vaccine, of value for children older than 2 years who are highly susceptible to pneumococcal infections, is seldom used in Britain at present. Varicella vaccine is undergoing clinical trials in the United States at present and has been used extensively in Japan[7] and might again be of benefit to susceptible children.

This brief survey of our current vaccination practices suggests that it is time that some recent advances were put to good effect in Britain. The policy of the DHSS has changed little in the past decade, and this complacency is detrimental to the health both of living children and children as yet unborn.

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Antidepressant effects of electroconvulsive therapy: current or seizure?

Nearly 20 years ago two large multicentre trials[1 2] established that electroconvulsive therapy (ECT) was superior to placebo tablets in the treatment of endogenous depression. These findings, together with the work of Cronholm and Ottosson[3] apparently showing that attenuation of ECT induced seizures with lidocaine reduced their antidepressant effects, seemed to establish that ECT was effective in depression and that its mechanism of action depended on inducing seizures. In recent years, however, this consensus has been undermined. Increasing public concern may have been responsible for renewed interest in the part played by the electric current and that played by the induction of seizures in the antidepressant efficacy of ECT.

Since 1978 four studies have compared courses of real versus simulated ECT (anaesthesia only) in depression. The results ranged from no difference in efficacy[4] to the striking superiority of real over simulated treatment reported by West—a study described as "double blind" by its single author.[5] Between these extremes, the Northwick Park trial reported a small and short lasting superiority of real ECT[6] and results from Edinburgh claimed a beneficial effect of real over simulated detectable after only two treatments.[7]

No satisfactory explanation for these discrepant findings has emerged. Much attention has focused on explaining the small differences between real and simulated ECT observed in the Lambourn and Gill[4] and Northwick Park trials.[6] Two main areas of criticism have been that the treatment was ineffective

and that the patients were in some way unsuitable. Despite the fact that convulsions were observed in every treatment in both trials, it has been suggested that night sedation with benzodiazepines may have attenuated seizures induced 11-12 hours later.[8 9] Few studies of ECT have controlled medication,[10] however, and cogent evidence will be required to substantiate the view that benzodiazepines should be withdrawn from all patients undergoing ECT.

Criticisms that patients were undertreated in some trials are based on the assumption that induction of brain seizures is the therapeutic agent in ECT and not the current itself. This assumption is based principally on the work of Cronholm and Ottosson, who claimed that currents above the threshold for inducing seizures did not improve the efficacy of the treatment, whereas attenuating the convulsion with lidocaine while keeping current constant did reduce efficacy.[3] The authors concluded that induction of seizure and not the current was responsible for the antidepressant efficacy of ECT. Methodological defects (non-random allocation; re-entry of patients) have been pointed out[11] in this comprehensive study; but more importantly, however, some challenging new findings now suggest quite different conclusions.

Robin and De Tissera[12] compared the antidepressant efficacy of three types of electrical stimulation, all of which were sufficient to induce convulsions of similar duration but which varied in waveform and the amounts of energy delivered. Adequate precautions were taken to keep raters and psychiatrists "blind" to the type of treatment each patient received. The results seem clear cut. Fewer treatments were required to achieve satisfactory results when intermediate or high energy stimulation was used than when low energy shocks were used and several patients in the lower energy treatment group failed to respond after nine seizures. Robin and De Tissera concluded that the amount of energy used to induce seizures has an important influence on the therapeutic outcome of ECT. The findings suggest that seizures may be an inevitable consequence of adequate therapeutic electrical stimulation but that seizures are not causally related to the therapeutic response. This conclusion shows how ignorant we still are of the mechanism of action of the treatment and throws into confusion attempts to define optimal electrical characteristics of stimuli to be used. Hence attempts to induce seizures with minimal, individually titrated doses of electrical energy may prove to be misguided.[13]

Robin and De Tissera point out that their ineffective low energy treatment (waveform IV on the Ectron mark IV machine) was used (unilaterally) in Lambourn and Gill's trial, which also found the treatment ineffective. In contrast, trials finding real ECT superior to simulated have used intermediate (waveform I, Ectron machine[6 7]) or high energy pulses (Transpsychon machine[5]). The major difference between these trials, however, is not the degree to which patients having real treatment improve, which appears quite similar, but the degree of improvement shown after simulated treatment, which seems to have been much greater in the Northwick Park[6] and Lambourn and Gill[4] trials than in the Edinburgh[7] and West trials.[5] Clearly differences in response to simulated ECT cannot be explained by the electrical characteristics of real treatments.

Perhaps of more relevance to the different results of these trials are suggestions that patients showing considerable improvements after simulated ECT would not normally have received electrical treatment. Yet in Lambourn and Gill's[4] trial 60% of the patients had previously received ECT. Fewer of the Northwick Park patients had received ECT, but patients had to fulfil two sets of entry criteria previously shown to select a group of patients responding to full ECT. The conclusion seems inescapable: whether selected for treatment on the basis of clinicians' judgment or of widely accepted objective criteria the condition of many patients appreciably improves with simulated treatment and gains little additional benefit from the passage of electricity. The West and Edinburgh trials suggest, however, that some clinicians can select patients responsive to the electricity and not to anaesthesia alone; if that is so the basis of their judgment remains unknown. Detailed analysis of the results of the Northwick Park trial suggests that the beneficial effects of electricity are confined to those patients with depressive delusions,[14] and this bears out earlier reports suggesting that deluded depressives respond better to ECT than to drugs.[15] The differences in the proportions of deluded depressives in the trials may go some way to explaining their different results.

In conclusion, the passage of electricity during ECT clearly does have antidepressant effects. Nevertheless, Robin and De Tissera's recent paper suggesting that seizures may not be causally related to the therapeutic effects of the electricity leaves the definition of the optimal stimulus values open. Even the clinical characteristics of patients who will respond to electricity remain uncertain, though deluded depressives may be particularly responsive. Despite these uncertainties, and in view of the safety of this treatment, there are probably no grounds for withholding ECT from patients with life threatening depression or with an unsatisfactory response to antidepressant medication.

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